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THE NATURE AND CAUSES OF

DUODENAL INDIGESTION

BEING THE

BRADSHAW LECTURE

DELIVERED BEFORE

The Royal College of Physicians

BY

W. H. ALLCHIN, M.B. Lond.

Fellow of the College.

Physician and Joint Lecturer on the Principles and Practice of Medicine to the Westminster Hospital, Examiner in Medicine to the Examining Board for England,

On NOVEMBER 26, 1891.

London
JOHN BALE & SONS
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THE NATURE AND CAUSES OF DUODENAL INDIGESTION.

Mr. President, Fellows of the Royal College of Physicians, and Gentlemen,—The series of lectures of which I am about to deliver to you the eleventh owes its name and origin to a bequest to this College made in the year 1880 by the late Mrs. Bradshaw in honour of the memory of her deceased husband, Dr. William Woode Bradshaw, some time extra-licentiate and member of this College, who died at Reading on August 18th, 1866. The testator stipulated that the income of the bequest should be "yearly expended in establishing and permanently maintaining an annual lecture to be de-

livered in connection with this College by a lecturer to be chosen by the President on some subject connected with physic." A similar bequest with a like object was made to the Royal College of Surgeons of England. It was further enjoined that the lecture should be delivered on August 18th, the anniversary of Dr. Bradshaw's death. So far as this College is concerned this condition has been faithfully observed during the past ten years; but the serious inconvenience attached thereto, together with the great probability that the primary object of the testator would thereby be considerably defeated, have induced the College to apply to the Charity Commissioners for permission to leave the selection of the date in the hands of the President. The consent of the Commissioners having been obtained, an alteration, more, it is hoped, in accordance with the convenience of the Fellows, is made for the first time this year. I think it altogether fitting that my first words should be devoted

to the commemoration of our benefactor, alike in honour of his memory and the encouragement of others similarly to benefit our College for the good of our profession and the increase of our common knowledge; and this I do with peculiar pleasure inasmuch as Dr. Bradshaw received his medical education in part at the medical school with which I have the honour to be connected.

In proceeding to determine in what way I could most profitably employ the opportunity offered to me, I was obliged to consider what subject I could best treat with any degree of completeness within the limits of my allotted time, and also what I might best pretend to speak on, I will not say with authority, but perhaps with some more intimate knowledge due to a familiarity with the questions involved. The one I have selected has long possessed for me exceptional interest, and towards it I have for some time especially directed my attention. Yet no one is more fully conscious than myself of the risk of failure that I court;

for apart from great inherent difficulties in the subject itself—difficulties which our present want of physiological knowledge renders insuperable—there remains the chance, if not the certainty, that much of what I may say, and perhaps in my own mind venture to claim credit for, may be well known to many in this learned company. I must rest content, therefore, if I should succeed in placing before you, in a manner that is comprehensive and yet suggestive of fresh inquiry and thought, a subject well deserving of your consideration.

The nature and causes of duodenal indigestion have not, so far as I have been able to ascertain, received at the hands of authors any very special attention. As a phase of the general processes of dyspepsia, it doubtless has found a place; but whilst various remedies have been suggested to relieve assumed imperfections in the performance of the function, I can find no detailed description of the symptoms referable to this disorder, or of the causes leading thereto, still

less any account of the grounds upon which its diagnosis may be based. I will even go further, and express a belief that there exists a general vagueness of thought and expression in respect to this subject far beyond what our present unavoidable ignorance would justify. The whole subject of indigestion, moreover, has, I venture to say, been far from being always considered in a scientific manner—in a manner, that is, based on physiological grounds. Perversions of other functions of the body have been of recent years studied with far greater regard to proved physiological data than have imperfections of digestion, which have furnished a happy hunting-ground for the impostor and the quack. One has only to read even in the works of those who are accounted among leaders to recognise a sort of accepted hopelessness in dealing with the problems of indigestion. The multiplicity and frequent remoteness of the symptoms are considered a reproach, and, except for

being occasionally treated of separately, are but seldom regarded from the point of view of the organs and functions primarily involved. The great frequency of dyspeptic troubles, and the general familiarity of the profession no less than the laity with certain symptoms thereof, have not engendered a corresponding accuracy in their study or rational basis of treatment. On the contrary, there are few groups of diseases which can show a more reckless empiricism in this latter respect. I fully believe that not a little of the obscurity attaching to the subject and the general vagueness of idea concerning it have a very distinct connection with a want of precision in the use of the terms employed, and with a lack of definition of the limits of the question, as well as a failure to recognise the exact relationship that the function of digestion bears to the entire economy of the organism. I find it needful therefore to preface my remarks on the proper subject of my lecture with some

preliminary observations of a general character.

Indigestion, or its synonym, dyspepsia, should be taken to signify some imperfection in the performance of the function of digestion, and should not, I hold, be made to include disorders of other functions clearly distinct from digestion, as is too often the case. The process of digestion is strictly concerned with the conversion of the solid and fluid ingesta into a fluid and diffusible form; it is essentially of a physico-chemical character, capable to a great extent of being imitated in our test-tubes and laboratories. It is, I think, most undesirable to extend the meaning of the term so as to include the process of absorption, still less those subsequent metabolic changes which tend to fit the digesta for their subsequent use in the nutrition of the tissues. These latter functions are wholly different from the acts comprised in digestion, and though equally if not more important for the well-being of the individual, are nevertheless performed by entirely different structures, and

depend on entirely different conditions. It may be urged, no doubt, that as digestion is a necessary preliminary to absorption, as this is in turn to assimilation, that for practical purposes they may be considered as one, especially as it is quite possible that some molecular changes in the ingesta are effected during digestion which are steps in the process of metabolism, apart from those alterations distinctly tending to the diffusibility of the materials. But I entirely demur to this. These three phases of the entire function of nutrition, whilst intimately associated and consequential, are, nevertheless, in the main distinct in the character of the tissues concerned no less than in the nature of the very acts themselves, and for an investigation of one or other of these functions when performed abnormally, for recognising the distinctive symptoms of each, the causes which lead thereto, and for proposing rational remedies, no limitation, I believe, can be too defined at first, fully appreciating, of course, the inter-

dependence of these functions among themselves and with all other functions of the body. If from among what have been designated the protean symptoms of indigestion we are enabled to discriminate those which owe their origin essentially to a flaw in the physico-chemical changes in the food from those which depend on a failure in the due absorption of the digested products, and still more from those caused by subsequent mal-assimilation, an important step has been made ; and there is no more reason why such distinction should not be drawn than there is for not making a differential diagnosis of the disorders of other functions. The expression "digestion by the tissues or interstitial digestion" merely denotes the anabolic processes of the tissues, and such extension of terms leads to no practical good, since the digestion by the tissues is doubtfully comparable to the processes carried on in the alimentary canal, and is wholly different in the conditions under which it is performed. It is worth while also

to remember that the real significance of the phrase "disorders of digestion" is very different from that of "diseases of the digestive organs." The greater number of the maladies which engage our attention are named and primarily regarded from an anatomical standpoint; the recognisable modification in structure, whether gross or minute, is made the basis of enquiry, and even when the disease is studied clinically it is with a more or less expressed reference to the associated or causative structural perversions. The nomenclature of our diseases is also mainly anatomical; and whilst I regard it as all-important to make anatomy the foundation of pathology, such a view does not meet all the requirements of the practical physician, nor completely satisfy our conceptions as to the real nature of morbid phenomena.

Whilst, therefore, by "diseases of the digestive organs" is signified those structural changes in the tissues of the organs expressed

by such terms as inflammation, degeneration, new growths, &c., and specifically denoted by such words as gastritis, enteritis, cancer of the liver, &c., comparable to similar diseases of the heart, lungs, or nervous system (endocarditis, pneumonia, cerebral abscess, &c.), the phrase "disorders of digestion" denotes imperfection in the functional performances of the digestive organs, with less implied reference to structural changes of those organs than is the case with disorders of other parts of the body. How seldom comparatively speaking, do we hear of cases of gastritis, or enteritis, of gastric cirrhosis or atrophy of the intestine, and how often of indigestion or dyspepsia, which are none the less immediately due, in the majority of cases, to structural perversions in the organs concerned. The fact is that in regard to digestive disorders we are far more in the habit of regarding the functions apart from the organs than we are in the case of any other regions of the body, and, as a con-

sequence, to place the disturbance of the function of digestion on a somewhat different platform to what we do disorders of other parts. In other words, the structural diseases of the alimentary tract and appendages are described and regarded apart from their relation to the digestive function. The symptoms of a gastritis or hepatic cirrhosis overshadow the true indigestion effects to which they give rise, and as a consequence we find ourselves insensibly separating tangible diseases of the alimentary organs from the symptomatic perversions of the digestive process. That such should not tend to elucidation, or to a clear conception of indigestion, must, I think, be admitted. It introduces an element of confusion fatal to a proper appreciation of what indigestion is, still more to the differential diagnosis of the various forms of the malady. I believe this to be in great part attributable to the fact that the function of digestion bears a somewhat different relation to the

whole organism than do the other functions of the body. I have described it as being essentially physico-chemical; that is to say, the preparation of the food for absorption, irrespective of its nutritive value and only conditioned by its digestibility, is affected by certain movements which bring about a finer subdivision of the food and a due mixing with certain solvent juices which convert the ingesta into a soluble and diffusible state. To a great extent these processes are but continuations of what is accomplished by the ordinary preparation of our food for the table. It is true that the movements of mastication, deglutition, and peristalsis are effected by a characteristically living tissue-muscle, but the results obtained are in no way different from what a mincing machine and a mixer can perform. The changes which are brought about by the various alimentary secretions, saliva, gastric and pancreatic juices, and bile, can be very closely imitated out of the body,

and their sole peculiarity is the presence in some of them of peculiar ferment bodies which depend for their formation on living protoplasm; and with their action digestion proper ends, whilst the subsequent absorption and metabolism are intimately dependent, more especially the latter, on vital activity. Moreover, the process of digestion is to all intents and purposes carried on outside the body on an epithelial surface, the lining of a canal which is open to the exterior at each end, and is in no sense within the body proper. It is only after the food has been digested that it actually gains entrance into the tissues by absorption. The relationship of the food during digestion to the tissue elements is therefore most remote; the various digestive secretions formed by certain specialised epithelia are thrown out by the cells upon a free surface, where they exert their characteristic effects upon the food constituents. A very considerable re-absorption doubtless takes place of the juices, but this would seem to be a matter of economy.

So long as the various stages of the digestive process are performed according to the standard which we assume as normal, unaccompanied by pain, and, indeed, for the most part without sensation, except, perhaps, of a mildly pleasurable kind, the function is to be regarded as healthy, whatever failure may occur in the absorption of the digesta and in their subsequent assimilation. Doubtless it will be urged that should either of these functions be imperfectly performed, the process of digestion itself will suffer; that delay in absorption of the digesta is a definite cause of dyspepsia, and mal-assimilation leads to a general mal-nutrition, in which digestion fails, along with other functions. True, of course; but I am contending that if we are to form a clear conception of what indigestion is as a preliminary to diagnosing what phase of digestion is imperfect, we must commence by having a clear notion of what digestion is strictly concerned with.

There is another characteristic of the function of digestion which requires consideration in view of the special subject of my remarks, and that is what may be termed its progressive character. Owing to the different nature of the alimentary principles and the corresponding different nature of the means for digesting them, they are not all reduced to a fluid and absorbable state at once, but at successive stages and in successive parts of the canal. Thus we have buccal, gastric, and intestinal indigestion ; and an important question arises : To what extent is the process, as carried out in later stages, influenced by or dependent on those which have preceded them ? Looking to the three groups of alimentary principles which require digestion—proteids, carbo-hydrates, and fats—the others being diffusible without change, the first-mentioned, and in one sense most important, element of food requires the greatest amount of digestion, and occupies by far the longest

time in so doing. The sequence in the digestion of these substances is also noticeable. Thus, commencing with the mouth, a considerable amount of the starch food is converted by saliva during mastication into soluble maltose, and for a short period this conversion is continued after the food reaches the stomach. In the stomach the proteids are mostly converted into diffusible peptones, a process which occupies a relatively long period. Up to the passage of the chyme into the duodenum the fats have been untouched, and are not unfrequently a hindrance to the due digestion of the other materials. First the carbo-hydrates alone, then the proteids alone, are acted upon, and there is little doubt but that some of the digesta are absorbed from the stomach as dextrose, peptones, and salts, whilst the greater bulk, which constitutes the chyme, passes on through the pylorus into the intestine for subsequent treatment, where the facilities for absorption are much

greater. The chyme, then, as it leaves the stomach, being emitted through the relaxed pyloric orifice at intervals during gastric digestion, appears as a grey grumous fluid of strongly acid reaction, and consists of such proteids and carbo-hydrates as may have escaped any treatment by the gastric juice and saliva respectively, products of the digestion of these substances not absorbed —namely, albumoses and peptones, dextrine and maltose, fats which have as yet received no treatment, unabsorbed salts and water, such indigestible substances as may happen to be present, and remnants of saliva with a variable quantity of the gastric juice. The passage of this material over the common orifice of the bile and pancreatic ducts excites a flow of these fluids, which arrest the action of the pepsin and finally destroy it, and, in virtue of their strong alkalinity, very quickly neutralise the acidity of the chyme, which in the lower part of the duodenum and throughout the greater

part of the small intestine remains alkaline. Briefly stated, the changes effected in the constituents of the chyme to further fit them for absorption by the villi depend upon the maintenance of a temperature of 100° F., or thereabouts, which, so long as life exists, may be said to be provided, and any known variations in which are not recognised as modifying the digestive process in any way; and, secondly, upon a sufficient supply of the bile and pancreatic juice, disregarding the succus entericus, concerning the action of which much uncertainty prevails. Practically what is for shortness termed "duodenal digestion" is the digestion of the chyme by these two fluids, which takes place throughout an unknown and probably variable length of the jejunum, and consists in a special preparation of fats for absorption, and in a supplementary treatment of carbo-hydrates and proteids which have each received individual digestion in the mouth and stomach. At the outset, then, intestinal

digestion, and more particularly that which may be denominated duodenal, is in marked contrast to the buccal and gastric, inasmuch as here for the first time all three alimentary principles are submitted to simultaneous digestion, and the opportunities for mal-performance would appear to be proportionately increased.

As a digestive agent—*i.e.*, as a solvent fluid—the bile is of little account, since it is very doubtful whether it exerts any effect by itself upon the proteids or carbo-hydrates, and as an emulsioniser of fats it is far inferior to the pancreatic juice, to which it appears to be distinctly accessory in its digestive action. But whilst occupying this secondary position as regards the conversion of the ingesta to a diffusible state, it has a very special value in two other directions, one of which especially concerns us here. First, it undoubtedly facilitates the *absorption* of fats, rendering the mucous surface more permeable; and secondly, it exerts a very definite action upon

the chyme, which appears to be a desirable preliminary to the pancreatic digestion. The proteids of the chyme, as already said, consist of those which have escaped peptic digestion, together with peptones and intermediate bodies known as acid albumen and albumoses. The peptones, which are both soluble and diffusible, are untouched, but the undigested proteids in solution and the acid albumens and albumoses, as well as the pepsin, are precipitated in a flocculent and granular form by the alkalies and salts of the bile. The exact object of this is not very apparent, but it is upon these substances that the trypsin ferment of the pancreatic juice exerts its action, with the result of converting them into diffusible peptones, similar if not identical with the products of peptic digestion, and through similar intermediary stages. The pancreatic digestion of proteids is, however, carried further in the disintegration of the molecules than the gastric digestion is, since the hemi-peptones of tryptic fermentation

may be converted into leucin and tyrosin, preliminary stages to the excretion of the nitrogenous excess as urea. How far this actually takes place in the intestine probably depends in great part upon the amount of proteids present. The conversion of the undigested starches of the chyme by the amylotic ferment of the pancreatic juice is exactly comparable to the action of the ptyalin of the saliva, perhaps aided therein by the bile, and the fats are chiefly emulsionised, and to a slight degree saponified. The salts and other diffusible elements of the chyme are absorbed from the intestine without further treatment.

Over and above the changes exerted by the digestive juices upon the intestinal contents, another agent has to be considered. How far it is effective in normal digestion is doubtful, but it very readily asserts itself, and becomes responsible for many indigestion results. I refer to the action of micro-organisms, various species of which normally

inhabit different regions of the alimentary canal, the mouth, stomach, and different parts of the intestine. Under ordinary circumstances they take no part in buccal digestion, and to only a very slight extent in the acid medium of the stomach ; but in the alkaline contents of the intestine they find a most suitable field for their activities and development. In normal conditions the changes which these bodies produce, in the small intestine at least, are scarcely, if at all, of a putrefactive character, and are probably in great part limited to a lactic and butyric acid fermentation of carbo-hydrates, a fat-splitting action comparable to that exerted by the steapsin of the pancreatic juice and a slight peptonising action on proteids. But it most certainly appears that none of these actions are essential, and so far as is at present known, the entire process of digestion would equally well be carried out in the complete absence of these organisms by the sole agency of the various juices. Their presence,

moreover, is to be regarded as a risk, since it would seem that the normal limits within which their action is kept, perhaps by the bile, is easily overstepped, with the result of giving rise to putrefactive decomposition of a serious nature, which, under ordinary circumstances, only occurs to a very slight extent in the large intestine and lower end of the ileum—that is to say, beyond the limits of action of the pancreatic juice, where it is believed that the intestinal contents, having lost their nutritive constituents by absorption, consist only of indigestible residue. In this connection I would quote from Professor Halliburton's recent valuable work on "Physiological Chemistry," the following suggestive remarks:—"A useful function fulfilled by the organisms appears to be the destruction of poisonous substances, such as cholin, the alkaloid derived from lecithin. It is possible that, if other alkaloids (leucomaines) are formed by the processes occurring in the intestines, these also are destroyed, for they are absent in the normal excretions."

Very briefly, but as necessarily preliminary to my subject proper, have I sketched the main features of intestinal digestion, or at least that part of it which may be conveniently denominated duodenal. I would specially emphasise what I have termed the progressive character of the process. The various alimentary principles are dealt with *seriatim* and then simultaneously, and it behoves us to try to ascertain how far the earlier steps in the process are essential to the proper performance of the later ones; whether, that is to say, buccal and gastric digestion, apart from the direct conversion they effect in certain food elements, are necessary to the subsequent satisfactory action of the duodenal secretions. In the experimental inquiries upon which much of our knowledge of intestinal digestion is based, I cannot help thinking this has not been sufficiently taken into consideration, and that untouched food stuffs have been submitted to the action of the pancreatic juice under

suitable conditions, but without any attempt to imitate the preliminary gastric treatment of them. I had myself partially devised some experiments keeping this object in view, and much regret that I have been unable to carry them out up to the present time, but hope to do so very shortly with the facilities offered by our laboratories on the Embankment. It is true that in animals the introduction of the crude foods through duodenal fistulæ has been followed by a complete digestion of them, the effect of the stomach being thus eliminated; but I am not disposed myself to regard the results as conclusive nor as fairly comparable to what takes place in the human subject. So far as I am aware, the only case in man that offers opportunity for observation on this point occurred at Guy's Hospital in the practice of Mr. Golding-Bird, and was reported by him in the nineteenth volume of the Clinical Society's Transactions. In a patient whose jejunum had been opened about four inches below the duodenum—exactly, that

is, where the biliary and pancreatic digestion is in progress—it was noticed that certain indigestion symptoms followed the introduction, through the fistula of such food as beef-tea, milk, bread, and corn-flour—*i.e.*, food which had not received previous buccal or gastric treatment. The case serves to illustrate another point, and I shall refer to it again, but so far as it goes it confirms what I am insisting on, viz., the all-importance of salivary and peptic digestion of the food before the bile and pancreatic juice are called upon to exercise their activity.

So much for the normal process of intestinal digestion. Now let us turn to consider in what way this may be imperfectly performed, and by what symptoms, if any, such imperfection may be recognised ; the evidences, that is, of the chyme not being so treated as to fit its constituents for absorption, at which point the digestive process proper ends. For clearness of description the conditions leading to such indigestion may be

summarised thus:—1. Some impropriety in quantity or composition of the chyme, including initial errors in diet. 2. Some perversion in the quantity or quality of the bile and pancreatic juice, more particularly the latter. 3. Some accessory fermentation process or processes of an abnormal character. 4. Exaggerated or deficient peristalsis. 5. Defective absorption of the digesta. Any form of duodenal dyspepsia must be directly referable to one or other of these causes. Is it possible to discriminate or to recognise any symptoms which may be clearly traceable to either of these states? It will be well, in any attempt to answer this question, to consider these groups of causal conditions somewhat in detail. A clear idea of the possible circumstances which may lead to imperfect digestion in the intestine should be helpful towards making the diagnosis.

First, as to the character of the chyme, the material to be digested and rendered absorbable. Obviously the perfectness of its di-

gestion may be as dependent upon its intrinsic nature as upon the efficacy of the agents by which it is to be acted upon. If the constituents of the chyme be impossible of digestion by the pancreatic juice and bile, the digestive result will be proportionately small. All our food of course contains a certain proportion of such material, which, passing along unchanged, contributes to the formation of the fæces. Cases where the great bulk of the food consists of such indigestible material are so exceptional and so easily corrected that further consideration of them need not detain us. In another class of diets there are few or many substances which are noxious, though perfectly digestible—digestible, that is, though not nutritious, a distinction not always sufficiently observed—the former is strictly what may be made in the alimentary canal diffusible; the latter is only what is of value in tissue metabolism, subsequent to absorption from the canal. In the greater number of cases it will be found, I believe,

that such substances receive fitting treatment in the stomach, whether by causing vomiting and so removal, or are rendered innocuous by the action of the gastric juice, and cannot be recognised as causing difficulty in the intestine. Exceptions of course do occur, and the toxic bodies, passing on into the bowel, there set up effects directed to their removal, by causing diarrhœa, often with pain and other symptoms. Such ingredients of the food are, however, accidental, and their presence, except for completeness of our subject, scarce require mention as a cause of intestinal dyspepsia. The conclusions arrived at by Sir Wm. Roberts respecting the effect of food accessories—such as alcoholic drinks, tea, coffee, beef-tea, and whey—upon salivary, gastric, and pancreatic digestion, are of exceeding interest, especially in reference to my present point. He found their general tendency was to retard digestion in the mouth and stomach in proportion to their strength, but that with regard to pancreatic digestion their effects were practically *nil.*

Very different, however, is it with those modifications of diet which consist in excesses or deficiences of those constituents which in themselves, and in proper quantity, are requisite, viz., proteids, fats, and carbo-hydrates, and, to a much less degree, salts and water. Insufficiency of the amount of any of the required alimentary principles, it may be said at once, shows itself rather in remote nutritive disturbances than in immediate digestive trouble, but excess in the quantity of one or more of these substances distinctly does, in the great majority of cases, directly bring about a dyspepsia more or less considerable. Doubtless here, as in so many other cases, habit does much, and the person who can digest anything can also usually digest any quantity which his appetite allows of his consuming. Excess of proteid ingesta, so far as it affects the quality of the chyme and the consequent duodenal digestion, will likely lead to a larger amount escaping gastric treatment than is normally the case, and the greater

demand, therefore, for treatment by the pancreatic juice. Here, again, in the majority of persons, the excess over what is actually required is probably met by an advanced stage of disintegration, with the production of large quantities of leucin and tyrosin, later, no doubt, converted into urea and its allies, and thus may become responsible for disorders primarily of metabolism or excretion, the work of digestion, so far as they are concerned, being properly performed, and there being no dyspepsia, at least of a primary character. Again, chyme containing a large excess of proteid matter, in various forms—more, we may fairly suppose, than the pancreatic juice can satisfactorily deal with—gives at once the opportunity for increased activity and development of those intestinal micro-organisms which are ever ready to assert their powers, and with this grave disadvantage, that whilst the trypsin fermentation ceases with the production of such comparatively harmless bodies as leucin and

tyrosin, the decomposition effected by the living germs becomes more and more putrefactive in character, and leads to the production of many toxic substances, leucomaines, &c., which, becoming absorbed, are largely responsible for the remote symptoms of dyspepsia to be afterwards considered. In the sense, then, of permitting excessive micro-organism activity, with its attendant risks, an overplus of proteid food becomes a cause, and one that is to be regarded as frequent, of duodenal indigestion.

The excessive ingestion of carbo-hydrate food, whether as starch or in the form of sugar, is liable seriously to modify the progress of digestion in the stomach, and thus to affect the character of the chyme. There is good reason to believe that in the earliest stage of normal gastric digestion the prevailing acid in the process is lactic, derived mainly from the carbo-hydrate food. Gradually this is replaced by hydrochloric acid, which alone is found after the middle of the

period occupied by the digestion of an ordinary meal. Should the source of the lactic acid be unduly excessive, the predominance of the hydrochloric acid is likely to be delayed, and perhaps not attained, with the result of very considerably interfering with the proper digestion of the proteids in the stomach, and their consequent appearance in the chyme, ill prepared for tryptic digestion. Moreover, when lactic acid is formed in the stomach to any considerable extent, as it is likely to be when there is very much sugar present, further organic fermentations are set up, with the production of various acids, especially butyric, which escape in the chyme, and are probably a further source of disarrangement of the intestinal digestion. Somewhat in contrast to the previously mentioned condition—viz., excessive proteid food, where the ill effects, so far as digestion is concerned, are produced in the intestine—here, where carbo-hydrates are in excess, the dyspeptic results are gastric in situation, or at least are so primarily.

Lastly, an excess of fat in the diet. This to a great extent exerts an ill effect by its mechanical distribution in the food. If it be in separate portions there is no reason that I am aware of to attribute to it any harm; but if it be presented as an oily coating to particles of proteid or carbo-hydrate, it protects these latter from the solvent action of the saliva and gastric juice until their escape in the chyme to the intestine, where the fat first receives treatment by the bile and pancreatic juice. Inasmuch as the various fats of food, in whatever form taken, are melted in the stomach, a large quantity might tend to prevent in this merely mechanical manner the due digestion of the other food constituents, and thus to determine a gastric, if not a subsequent duodenal indigestion of these substances.

I have so far spoken of dietetic errors in the main groups of alimentary principles as they may occur singly, but it is, of course, often the case that two or more of these im-

proprieties coexist, with, it may be presumed, a corresponding complexity in dyspeptic results. The interactions, if any, which take place among—or, at least, the influences exerted on each other by—the different food stuffs in the progress of their digestion are but little known in health, and still less in the course of disease.

The chief imperfections in the chyme, then, that we can fairly assume to exist, exclusive of noxious substances occurring in the food, are a large excess of proteids very partially and deficiently treated by the gastric juice, and a very considerable excess of lactic, butyric, and other acids, the results of carbohydrate decomposition. Both derangements may occur together, and the former certainly in the way shown, and possibly also the latter may be the cause of mal-digestion in the intestines.

Supposing, however, the diet be all that is satisfactory so far as it conforms to what is required in digestibility and composition—

and how often are the most careful and regular in their feeding severe sufferers from indigestion—there yet may be a very imperfect gastric digestion due to improper quantity or quality of the gastric secretion, or to some deficiency in the movements of the stomach, whereby the contents are not properly mixed or are not ejected into the duodenum as they should be, the whole process being thereby delayed, and giving rise to organic fermentations of the contents, as may be seen in an aggravated form in the ordinary state of gastroectasis. It is apart from my subject to discuss the causes of gastric indigestion ; suffice it to say that, whatever they may be, the resulting chyme which is submitted to duodenal digestion is impaired as regards the changes in its proteid elements, and may contain large amounts of lactic and butyric acids and other products of carbo-hydrate fermentation—similar results, that is, to those which may attend an excessive ingestion of either of these alimentary principles. Should both the

diet be improper and the gastric juice be deficient, it is reasonable to suppose a still further defect of the chyme in the absence of any compensations or adjustments which are at present quite unknown to exist. In these various directions, then, may we conceive of the chyme being abnormal, and as such the cause of duodenal indigestion.

Another cause for the same is to be found in the deteriorated quality of the duodenal secretions, or a deficiency in their quantity, which may happen with either a perfectly normal or a seriously vitiated chyme. Practically, as we have seen, the pancreatic juice is alone to be taken account of as a digestive agent, the bile being only accessory thereto. As regards the composition of the juice, we have only an uncertain knowledge, though occasionally opportunities have offered to obtain it from the main duct, and we certainly have no definite indications as to any modifications it may undergo in disease, such as we possess in respect to the amounts of the acid

and pepsine of the gastric juice; but its efficiency may be suspected to be impaired in general states of mal-nutrition, such as fever, anæmia, and the like. But if we know nothing of alterations in the composition we are not infrequently able to detect deficiencies or, indeed entire absence, of one or both of these secretions from the intestine. Occlusion of the main ducts by calculi or pressure of tumours will lead to this condition, and afford opportunity for the study of intestinal digestion in their absence. Deficiency or absence of these fluids signifies, of course, incomplete digestion of the chyme, and apart from the direct dyspepsia which such may cause, indirect effects may be induced by non-arrest of the peptic digestion which should normally take place, although this may be a beneficial compensation, by permitting a continuance of the proteid changes and peptone formation by the gastric juice, which otherwise the pancreatic fluid should have accomplished. In exclusion of these secretions

from the duodenum the precipitation of the albumoses above described can hardly be supposed to take place, perhaps again advantageous in the absence of the trypsin ferment. Sir William Roberts has shown, in contradiction to views that had been maintained, that the activity of the pancreatic juice is arrested by the acids of the stomach, and it is not improbable that in those conditions already described, where the chyme contains a large excess of the gastric acids, the power of the pancreatic juice is diminished or even temporarily destroyed.

A third group of conditions which are undoubted causes of intestinal indigestion, are those abnormal or excessive organic fermentations which have already been referred to as liable to follow a defect in the quality of the chyme. The bile is usually regarded as specially concerned in the prevention of these fermentations, or, at least, those of a putrefactive character, and entire absence of bile ought to be followed by considerable changes

of this nature in the intestinal contents. Yet my own experience would lead me to very much question the great power of the bile in this respect. At all events, it is no uncommon thing to find the stools absolutely free from bile, and yet with no specially putrid characteristics; whilst, on the other hand, those which may contain fully the normal quantity, so far as can be estimated by colour, may be remarkably offensive. I believe myself the putrefactive changes are mainly effected low down in the canal, and are not much influenced by the bile, and I am quite unaware of any control being exercised by this fluid over those toxic substances which may be formed in the intestine from the decomposition of the proteids.

Lastly, as causes of intestinal indigestion may be mentioned deficient or exaggerated peristalsis and insufficient absorption into the vessels of the villi, with consequent retention of the digesta in the canal. Perversions of peristalsis may be induced by the irritating

character of the chyme, causing diarrhœa, with mal-digestion and imperfect absorption; or the movements may be hindered by œdema of the muscular coat from congestion, by degeneration, by excessive distension or disturbed innervation. The delay in the onward progress of the chyme favours the activity of the micro-organisms present with the results already indicated, whilst deficient peristalsis means insufficient mixing of the food with the secretion, and also lessens the secretion itself, as well as hindering absorption, both of which the movements of the canal very considerably stimulate. But there is a special disorder of the movements of the stomach to which I am of opinion not a little indigestion is due—viz., an exaggeration of the normal propulsive movements, with undue relaxation of the pylorus, thereby facilitating exit of the gastric contents, especially the larger solid portions, before the peptic action is sufficiently advanced, and so, in the ways already referred to, interfering with pan-

creatic digestion. The opposite condition —namely, deficient gastric movements, with consequent retention of food in the stomach —has also to be mentioned, as causing an abnormal chyme and subsequent liability to duodenal dyspepsia. There is good reason to believe that if the diffusible products of digestion be not absorbed as they are formed, the continuance of digestion is unfavourably interfered with, whether in stomach or intestine, owing to putrefaction taking place in the substances retained ; and it would further seem that the due secretion of the various juices—certainly the gastric—is very directly dependent, among other things, upon the regular absorption of material from the canal.

To recapitulate the conditions which are immediately provocative of an indigestion in the upper part of the intestine, they are (1) Initial errors in diet ; (2) defects in the preparatory buccal and gastric changes ; (3) perversion of biliary or pancreatic digestion ;

(4) excessive and abnormal micro-organism activity; (5) exaggerated or deficient peristalsis; and (6) imperfect absorption of the already digested materials. It is probably rare that one of these causal conditions occurs alone; more commonly several contribute to the existent dyspepsia. It is to be understood that I speak here of immediate causes only. The circumstances which lead to them time does not allow me to touch upon, but it is of course implied that these conditions, in their turn, are the expressions of structural changes in the organs involved, whether these be as obvious as a gastritis, or as impossible of detection as a disturbed innervation. But it should not be forgotten that among the antecedent circumstances of these immediate causes is often to be reckoned dyspepsia itself, since the imperfect tissue changes thereby determined lead to an impaired structural condition of the digestive organs, with deteriorated functions and vitiated secretion of juices, thus maintaining a vicious

circle which intensifies itself. The dependence of indigestion also upon disorders of metabolism or on mal-excretion of tissue waste—errors of nutrition leading to the circulation of a vitiated blood, with consequent deterioration of glandular secretion—is too obvious to require more than mention.

Looking at the subject in the way I have done, the following questions suggest themselves, and though not capable of solution at present, are, I think, worth stating as helping to define the limits of our knowledge and to indicate the direction of future inquiry. Given a case of gastric dyspepsia, from whatever cause, with its special symptoms, must the resulting abnormal chyme determine a further duodenal dyspepsia with fresh symptoms, though other conditions in the bowel may be taken as satisfactory? Or may the duodenal process correct and compensate the stomach failure, with the result that the indigestion is restricted to the latter region? Or, again, in a case in

which there is no evidence of gastric indigestion, can a true duodenal dyspepsia arise from any local causes—deficiency of secretions, micro-organisms, &c.—producing symptoms, if not of a distinctive character, at least primarily attributable to it? Admitting that gastric indigestion may cause pancreatic indigestion, the two coexisting, may each occur independently and alone?

Passing now to the symptoms, are we able to discriminate any which especially betoken mal-digestion in the intestine as there are those which may be regarded as diagnostic of gastric dyspepsia? To answer this it will be requisite very briefly to consider the various symptoms referable to disordered digestion in general before proceeding to group them in respect to their immediate causation. For convenience of description of such numerous and varied phenomena, it is almost necessary to attempt some sort of classification, and the first and most obvious division is into those which

are clearly connected with the alimentary viscera—*i.e.*, local symptoms—and those which are remote and manifested through other than the digestive organs; an artificial distinction, I admit, and not capable of being rigidly drawn. Among the former or local symptoms will be included—1. Pain and all other sensory perversions, varying from a mere sense of weight at the epigastrium to acute, griping colic, or sensations of a peculiar character, such as heartburn. 2. Evidences of perverted peristalsis, such as diarrhœa, constipation, vomiting, with all varieties of ejecta from unaltered food to blood, bile, clear acid fluid, or yeasty fermenting material. 3. Signs of undue distension of parts of the canal, flatulence and meteorism, causing general abdominal fulness. 4. State of the tongue and mouth; salivation. 5. Character of the evacuations.

The remote symptoms of dyspepsia almost defy arrangement, and complete enumeration would occupy too long. Again pain

heads the list, in shoulders, back, or limbs; and, above all, headache, frontal, occipital, or vertex; as well as various neuralgias. The muscular system manifests a general weakness, lassitude, and distaste for exercise, which is badly borne; or there may be cramps, specially in the muscles of the calf. Affections of the circulation are of extreme frequency—such as flushings after food, cold extremities, palpitation, cardiac irregularity, fainting, and angina. The respiratory function exhibits such perversions as cough, dyspnœa, and hiccough; whilst vertigo, muscæ volitantes, subjective sensations of hearing or smell, all degrees of mental perversion, from a passing irritability to a lasting hypochondriasis, or apathy, drowsiness, or insomnia, and impaired appetite, may be conveniently set down as nervous manifestations, as well as pain already mentioned. Characteristic among the remote symptoms are skin eruptions, mainly of the erythematous type. And

finally may be mentioned abnormalities of the urine. It is not, of course, claimed that this list is complete or precise in arrangement, any more than it is suggested that those symptoms mentioned are all of equal frequency of occurrence. The extreme variety of combination is scarcely less remarkable than the multiplicity of the phenomena. Is it possible to select from among them any which may serve to localise the primary, or, may be, sole seat of the digestive failure? I fear only very partially, at least, so far as the duodenal digestion is concerned. The complexity of causation of most of these phenomena renders the unravelling of the problem well-nigh hopeless; but it is none the less desirable to set forth what may be affirmed with approximate certainty, remembering that at this point occurs the risk of confounding symptoms which are clearly due to defective tissue metabolism, with those which are purely dyspeptic. Attempts have been made

to refer dyspeptic phenomena to stomach or intestine, based on an observance of the time of their occurrence in respect to the taking of food. But a circumstance which goes far to vitiate the results is that both gastric and duodenal digestion progress to a great extent simultaneously ; for very soon after the food enters the stomach the propulsive movements of the viscus begin with the object of propelling the fluid portions into the duodenum, there to be submitted to the action of the bile and pancreatic juice ; and hence symptoms which may arise within three or four hours after taking an ordinary meal cannot with certainty, on the ground of time of their occurrence, be attributed to stomach or intestine. Nor could any greater probability be well asserted for symptoms occurring later, since many such are dependent upon long-delayed gastric digestion with deferred propulsion into the duodenum.

Certainly, I do not think we can find

among the local symptoms many that may be termed diagnostic of situation, however suggestive some may be; and the fact that not a few are equally associated with structural diseases of the canal itself, as well as with assumed imperfections of the digestive process taking place therein, adds much to the difficulty, more especially if both abnormalities coexist. The sense of weight and sinking at the epigastrium so often complained of, so far as we know its explanation, will equally be referable to the stomach or upper part of the intestine; but the time of its occurrence in relation to meals would usually suggest its greater frequency while most of the food is still in the stomach, and that it really does depend upon mere bulk of substance, with impaired movements of the organ. Mr. Golding-Bird's case of jejunostomy already referred to bears this out; for on introducing from fifteen to twenty ounces of food into the jejunum the patient immediately experienced a

feeling of sinking, or as he said, "faintness," with flushing of face, rapid pulse, and extreme discomfort. These symptoms were invariably repeated so long as the bulk of the meal remained as stated, but did not appear when the quantity of food given was much reduced; and from this circumstance Mr. Golding Bird suggested that these symptoms may be sometimes due in patients to a too rapid passage of the contents of the stomach through an unduly relaxed pylorus. But I think it would be difficult to discriminate between such, if such cases there be, and those where the same symptoms are apparently caused by too great a bulk of food in the stomach, and disappear on vomiting—too great, that is, for that particular stomach at that particular time, though not necessarily greater than may be taken at other times with impunity. Actual pain that may occur in the course of digestion is extremely obscure in cause, if we exclude irritating ingesta of all kinds and pain due to lesions of the canal,

which then becomes a symptom of definite disease of the organ, such as ulcer, and is not an evidence of disordered digestion proper. It is of course possible that during a faulty digestion acrid substances may be developed and produce pain as surely as similar materials do when swallowed in the food, and it is also probable that, if such do occur, they are formed during pancreatic digestion. But it is also probable that many cases of intestinal colic are of a spasmodic origin, expressions of some want of harmonious action between the nerves and muscles of the bowel, perhaps induced by the character of the contents; or, on the contrary, may be centric in origin. It has been asserted that pain in the region of the right scapula is indicative of duodenal disturbance, but I am not aware of any clinical evidence in support of the statement, and if it be true it is to be compared to a similar pain occurring in hepatic affections, and is referable to actual structural changes in the organ, rather than

to imperfect physico-chemical changes in the bowel contents. Nor can such irregularities as constipation or diarrhœa be made a ground upon which to determine the existence of a gastric rather than a duodenal indigestion, since it would be easy to show that failures in either situation might determine either of these conditions, just as in a large number of cases both depend on states of the lower part of the intestine beyond where the influence of the pancreatic juice may be supposed to extend, and therefore on conditions beyond the limits of my subject.

Some of the local symptoms I have enumerated are more or less significant of gastric disturbance, and gastric disturbance only, finding no explanation in intestinal states. Such are especially acidity, heartburn and pyrosis, all due to the presence, whether in excess or not is doubtful, of gastric acids, hydrochloric, lactic, or butyric, with possibly some hyperæsthesia of the mucous membrane. The occurrence of these symptoms, whilst un-

doubtedly pointing to stomach indigestion, does not of necessity exclude a coexistent duodenal failure. Somewhat less positive gastric symptoms are nausea and vomiting. Both may be due to causes quite remote from the digestive organs, and I have had reason sometimes to think that the former, if not both, have been the result of absorption of toxic intestinal digesta. But nevertheless, in the great majority of cases, these two symptoms are referable to stomach rather than bowel.

My experience so far has not led me to recognise any appearance of the tongue which can in any way be considered diagnostic of intestinal indigestion, even if there be any positively indicative of true gastric dyspepsia. General states, such as pyrexia or local conditions of the mouth and salivary flow, are, as Dr. Dickinson has shown, far more potent causes of morbid states of the tongue than disturbances in the functional performance of digestion lower down.

Setting aside, then, those phenomena which are either distinctly or mainly gastric in nature, or are indifferently evidence of stomach or intestinal disorder, there only remain to us for diagnostic purposes, among the local symptoms, flatulence and the character of the stools. Without doubt the former is, with certain reservations, to be regarded as a valuable diagnostic sign. But it is needful to eliminate those cases in which the excessive formation of gas is strictly limited to the stomach. Such cases are usually easy to detect; physical examination suffices to demonstrate them; but apart from the fact that the gastric distension may not always exceed normal limits, the excess of gas being eructated or passed on into the bowel, there still remain a number of cases in which there is reason to suppose that there is a regurgitation of carbonic acid from the duodenum into the stomach, as Sir William Roberts supposed, and it is probable that most gas of an ill-smelling character which is eructated originally

comes from the intestine. Speaking generally, however, over-distension of the bowels with flatus, especially if accompanied with much voidance per anum, is to be accounted as evidence of mal-digestion in the intestine, though even then it cannot be always set down to mal-performance within the scope of the duodenal processes, since fermentation changes, accompanied by formation of much gas, does also occur in the colon without any reason to suspect failure higher up. Sulphuretted hydrogen and marsh gas may be looked upon as exclusively developed in the intestine, but the latter may certainly originate in the large bowel as a decomposition product of its contents. But it is not all cases of true flatulence of the small intestine that are the results of an indigestion. There undoubtedly take place throughout the canal extensive gaseous interchanges between the blood in its vessels and the air it contains, and interference with this interchange may readily determine accumulations of gases in

the bowel independently of any defect in digestion. Otherwise, making due allowance in the directions I have indicated, intestinal flatulence is probably the most reliable local sign of duodenal dyspepsia, owing its origin to excessive ferment action of micro-organisms.

Neither the appearances presented by the stools nor their chemical investigation are as reliable as evidences of indigestion as they might reasonably be supposed to be, or as we may hope, with more extended knowledge, they will be. An excessively hurried passage of the ingesta along the canal may lead to much of it appearing in the evacuations unchanged, or almost so; but though such a condition would *de facto* indicate both gastric and intestinal indigestion, there would be nothing to show that the secretions were in any way at fault, and that it was anything beyond a much-increased peristalsis. Excepting such cases and the particles of food which have accidentally escaped digestion and

are always found in the fæces, the only aliment which passes unchanged and unabsorbed in any quantity, is fat—that is, the one which receives no treatment until it has escaped from the stomach. When the bile and pancreatic juice are excluded from the duodenum, this substance appears both unaltered and as crystals of fatty acids with soda, lime, and magnesia. Such motions as these often contain also a large quantity of peptones which are wanting in health. But this latter condition may be as much a sign of defective absorption as it is of imperfect digestion, though, so far as it goes, it, like the presence of fats, suggests the probability of improper duodenal changes. Of such bodies as ptomaines and other products of abnormal micro-organism activity, we know too little to allow of their occasional presence in the evacuations being useful for diagnostic purposes.

Before any attempt can be made to select from among the numerous and varied distal symptoms of indigestion those which may

serve to indicate duodenal failure, it is almost necessary to seek what explanation there may be for their occurrence. Their diversity, their interchangeability, their frequent rapid appearance and disappearance, no less than the intrinsic character of many of them, irresistibly suggest that in some way or other the nervous system is concerned. Some such, as pain, paræsthesia and subjective disorders of the senses, are clearly to be so explained; whilst others are obviously vaso-motor disturbances, as others are expressions of perverted trophic influence. Such phrases, I admit, do not carry us very far, but they represent our present way of thinking. There is also the question, How are these disordered nerve influences themselves originated? Are they reflex, determined by gastro-intestinal peripheral stimulation; or are they centric, induced by toxic agents absorbed from the canal and conveyed by the blood to the cortical "nervous arrangements," from which, in consequence,

they emanate and come to be expressed by the symptoms we speak of as dyspeptic? These are questions easier asked than answered, but they are framed entirely in accordance with our present conceptions.

Many of these symptoms may be caused by drugs taken into the stomach or injected subcutaneously, so that we have warrant for the suggestion of their poisonous origin, and certainly most of the vascular phenomena, if not others as well, are capable of being artificially produced reflexly by peripheral stimulation. It is not easy to suppose any other method of action from the gastro-intestinal tract than by the blood to the nerve centres, or by nervous irritation, but the supposition does not help very much to distinguish between gastric and upper intestinal situation ; the blood channels are the same, and the nerve paths take the same course. If it could be shown that these remote symptoms are wholly reflex in origin, I do not see how we could suggest any

grounds for referring any given symptom to stomach rather than to duodenum, or *vice versâ ;* but if the phenomena be toxic in cause, then probability would lead us to refer them to the intestine rather than higher up, since in that situation poisons are more likely to be developed. But, as I have more than once said, the responsibility for many of these symptoms must not be thrown on the digestive process. The assimilative power of the tissues themselves may be at fault, or between the actual results of the digestion and their final presentation to the tissue elements abnormal changes may take place in the pabulum in connection with the many protoplasmic influences which intervene.

Certain products of digestion, peptones and albumoses, as well as further results of proteid decomposition, such as indican, are of frequent occurrence in the urine. But the presence of the first-named is by no means associated with dyspepsia ; and

indeed, since the object of digestion is the formation of these bodies, their existence rather proves the excellence of the digestive powers, and their occurrence in the urine when derived from the alimentary canal would be due to some errors in their destination after digestion and absorption, much as glycosuria is. As regards indican and its allies of the ether-sulphuric acid series, it is probable that they may ultimately become valuable aids to the detection of abnormal splitting up of proteids in the alimentary canal under the influence of micro-organisms.

From this survey of the symptoms of indigestion the conclusion, I think, would be warranted that those which are capable of being specially referred for their causation to imperfect digestion in the upper part of the intestine, and that may be considered in any way diagnostic of such imperfection, are attributable to excessive, if not perverted, micro-organism activity—a condition which we have good reason to believe is quite a

superfluous accessory to the normal biliary and pancreatic changes, and one liable to be attended with much risk. A clinical point which to my mind has much significance in this connection is that in those not infrequent cases where the bile and pancreatic secretions are entirely excluded from the intestine, and when therefore a normal duodenal digestion is impossible, symptoms ordinarily described as dyspeptic are noticeably absent. Symptoms dependent on the structural disease causing the obstruction to the entrance of these fluids may of course exist, but few, if any, directly due to improper treatment of the ingesta. The patient will probably emaciate, and fat will occur in the stools, the digestive agents for that special alimentary principle being absent. But we look in vain, as a rule, for headache, palpitation, flushing, or even flatulence—that is, for those phenomena which perverted germ activity would produce. I am inclined to explain this by

the chyme from the stomach meeting with no neutralising fluids; the acids of the gastric juice pass on into the intestine and destroy the organisms which might otherwise have given rise to noxious fermentations. But be that as it may, the point I would urge is that complete absence of proper duodenal digestion may be unattended with dyspeptic symptoms, which goes far to support my contention that such symptoms are in the main due to imperfections of gastric action giving rise to primary phenomena of stomach indigestion; and secondarily, by providing a chyme that is vitiated, determining additional symptoms originating in the intestine, and mainly due to the action of micro-organisms which in normal circumstances would not have taken place.

Let me conclude with a few words by way of summary. In view of the object of my remarks—viz., the nature, causation, and diagnosis of duodenal dyspepsia—I have insisted on the necessity, for the proper

study of the subject, that a strict understanding should be come to on what is exactly the scope of digestion, since many symptoms referred to mal-performance of it are in reality due to perversions of functions —absorption and tissue metabolism—which do not commence until digestion has finished, and are wholly different in nature and conditions of exercise to the physico-chemical processes which take place in the alimentary canal. Also that the character of the successive digestive changes is such that interference with one is liable to seriously modify those which follow, and that this especially holds good in respect to the gastric digestion, which is a necessary preliminary to the duodenal, the normal performance of which is intimately dependent on a healthy chyme —far more, in fact, than upon the action of its own proper secretions. Further, that the most important factor of duodenal dyspepsia is its dependence on abnormal fermentations, caused by micro-organisms introduced *ab*

extra, which in healthy conditions do not assert themselves, and are wholly superfluous adjuncts to digestion. And, lastly, that any symptoms which may at all serve to localise the imperfect performance in the intestine are the results of these organic activities.

I do not pretend to have treated my subject exhaustively ; it were impossible in the time at my command. The entire question of treatment I have not been able to touch upon, though I will affirm my belief that most benefit is to be done in this direction by such diet or drugs as will correct gastric deficiencies, but I have attempted to map out the outlines and broad features and to lay down a course for the further study of an obscure and important subject in accordance with present physiological knowledge and the prevailing conceptions of pathology. It may be objected that in so doing I have, unnecessarily, if not unwarrantably, restricted the scope of the question, and have regarded digestion and its faults

too much apart from the influences of other functions, and that no such limitation as I have drawn can occur. But I feel sure that to obtain correct notions of the problems presented no limitation can be too definite at first; when they are acquired we may more profitably enter on the subject of other influences.

It only remains for me to express to you, Sir, my very deep appreciation of the honour you have done me in selecting me to address you to-day, and my warmest thanks to the Fellows in College assembled who approved your choice, and to hope that neither you, Sir, nor they have felt reason to regret the manner in which I have fulfilled the trust.

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